

Amendments to the Specification:

Please replace the paragraph beginning at page 1, line 5, with the following rewritten paragraph:

-- This application claims priority to U.S. Provisional Application 60/047,181 filed May 10 May, 20, 1997, now abandoned; PCT/US98/10318, filed May 20, 1998; and is a divisional of U.S. Serial No. 09/443,766 filed November 19, 1999.--

Please replace the paragraph beginning at page 1, line 13, with the following rewritten paragraph:

-- A major problem in breeding swine is to keep them disease-free. Intestinal disorders postweaning are a particular problem. A limited number of serotypes of toxigenic Escherichia (*E. coli* (*E. coli*) strains are the causative agents of oedema disease and postweaning diarrhea in swine which induce serious economic losses, especially among piglets aged 4 to 2 2 to 4 weeks, in swine breeding farms all over the world. The typical symptoms of oedema disease are neurological signs such as ataxia, convulsions and paralysis. At post mortem examination, oedema is typically present at characteristic sites such as eyelids and forehead, stomach wall and mesocolon. The diseases are caused by Shiga-like toxin-II variant and enterotoxins LT, Sta, Stb respectively, produced by *E. coli* that colonize the surface of the small intestine without effecting major morphological changes of the enterocytes (cells in the intestine). Certain types of bacterial *E. coli* strains, F18, F4 and K88 are major lethal villains in this regard. "Oedema disease of pigs is an enterotoxaemia characterized by generalized vascular damage. The latter is caused by a toxin, Shiga-like toxin II variant, produced by certain strains of *E. coli*" (Bertschinger *et al.*, 1993). The *E. coli* are distinguished by their pili types, a group of adhesive fimbriae that are related are designated *e.g.*, K88 or F18 (Vögeli *et al.*, 1997).--

Please replace the paragraph beginning at page 1, line 30, with the following rewritten paragraph:

-- Not all swine succumb to *E. coli* infections. Colonization depends on adherence of the bacteria to the enterocytes which is mediated by the bacterial fimbriae designated *e.g.*, K88 or F18. Susceptibility to adhesion, *i.e.* expression of receptors in swine for binding the fimbriae, has been shown to be genetically controlled by the host and is inherited as a dominant trait with,

in the case of F18, *B* being the susceptibility allele and *b* the resistance allele (Vögeli *et al.*, 1996; Meijerink *et al.*, 1996). The genetic locus for this *E. coli* F18-receptor (*ECF18R*) has been mapped to porcine chromosome 6 (SSC6), based on its close genetic linkage to the *S* locus and other loci of the halothane (HAL) linkage group on chromosome 6. The receptor for K88 *E. coli* is on chromosome 13. --

C3
Q&T

Please replace the paragraph beginning at page 5, line 15, with the following rewritten paragraph:

C4

-- An aspect of the invention is an isolated DNA molecule with a substitution of adenine for guanine in position 307. This molecule may also ~~be~~ have a substitution of adenine for guanine in position 857. Other isolated DNA molecules of the present invention include those with a mutation at nucleotide position 229 of the sequence of FIG. 1, wherein the codon CTT is changed to TTT, encoding for the amino acid phenylalanine instead of leucine. A mutation at nucleotide position 714 is from GAT → GAC, but there is no accompanying amino acid substitution in the encoded product. --

Please replace the paragraph beginning at page 5, line 24, with the following rewritten paragraph:

C5

-- A molecular assay for detecting *E. coli* F18 receptors in swine is to (a) isolate DNA from porcine nucleated cells; (b) amplify the DNA in a polymerase chain reaction (PCR) using oligonucleotides as primers which are complementary to a DNA sequence of the porcine alpha (1,2) fucosyltransferase gene 1; (c) perform a restriction enzyme digest with at least one restriction enzyme *e.g.*, CfoI; (d) separate the resulting fragments by gel electrophoresis; and (e) determine the respective numbers and lengths of fragments on the gel; and (f) determine from the numbers and length of fragments of F18, which receptors are present in the porcine cells. Use of the larger amplified fragments disclosed herein for restriction length polymorphism analysis (RFLP), rather than smaller fragments, is less expensive because the DNA bands can be run on agarose gels of relatively low concentration. Also, to produce some of the fragments, only one restriction enzyme is needed for a constant restriction site adjacent to the variable diagnostic site. --